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RESTORATION BY A NEW METHOD.

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THE CESSATION OF RESPIRATION UNDER CHLOROFORM,

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NOTWITHSTANDING the manifest tendency during recent years to employ, for purposes of anæsthesia, sulphuric ether instead of chloroform, it is obvious that the latter drug is with many surgeons still the favourite. So far as I can learn, Scottish surgeons, with few exceptions, employ chloroform, only turning to ether in special and comparatively rare cases. On the other hand, in England and America the case is becoming rapidly reversed, the majority of operations being performed under ether, and the exceptional ones under chloroform. This recent return to the older anæsthetic is to be explained by the growing impression that in point of safety ether has the advantage, while much of the difficulty of its administration is being overcome by improved apparatus and better methods of exhibition. Still, in spite of an admitted degree of greater safety, the administration of ether is undoubtedly attended by more than one disadvantage, and there is a suspicion, probably well founded, that all the risks attending its use do not end with its administration; but that to the list of inconvenient results must be added not a few pulmonary and other complications which may follow, and which may reasonably be attributed to its action.

Be this as it may, chloroform is possessed of many advantages, not the least of which is its easy administration and its quick action; and it will no doubt continue to be employed by many until at least some substance more satisfactory than any yet brought forward is met with. Certainly I should think that there are but few obstetricians who would be enticed from the use of chloroform by any of the advantages which may be claimed for ether in general surgery. The extreme inflammability of the vapour, and the bulky apparatus required for its satisfactory administration, are in themselves sufficient to prevent its general use

in this department. Moreover, all agree that for children chloroform rivals all other anæsthetics in the efficiency and safety of its action. Such being the case, it is probable that these advantages will secure for chloroform a very considerable employment in surgery and obstetrics, and will render its action a study of deep interest to the practical surgeon. On this ground I have ventured to bring before the Society an account of some observations I have been able to make, and which seem to me to involve points of some practical interest in relation to the physiological action of chloroform. I have no purpose of pleading the cause of one anæsthetic over the other. My object is merely to call attention to some features of the action of chloroform which seem to me to have been overlooked in previous investigations.

When we turn to the records of observations on the action of chloroform, we are struck with the amount of attention bestowed on the effect of its administration on the action of the heart and blood pressure, and with the careful records which have been made of this; while, at the same time, the effect on the respiration is indicated in comparatively loose terms, and graphic records are seldom given. It would seem, indeed, that while the potency of chloroform as a respiratory poison was recognised from the first, the difference of its action as compared with ether on the heart and circulation has engrossed the attention of observers. Thus it has come about that less attention has been paid to the respiratory changes brought about by its influence than to the remarkable effects produced on the circulatory system.

The records of these latter changes are ample and accurate. Thus the Glasgow Committee[1] concerned themselves almost wholly with the circulation, and give several important manometric tracings of blood pressure, but none of the respiration, during any of the experiments, and their account of the respiratory phenomena is by no means so full as it might be. And they are by no means singular in this respect, the same meagreness of detail being characteristic of other reports.

As my observations have shown me that the process of respiratory cessation in animals does not always present the same type, but is subject to variations of marked character, I have thought I might be justified, in view of this deficiency of existing records, in bringing these observations under notice.

My experiments were conducted on rabbits; and while I do not mean that we can justifiably reason directly from them to other animals, yet it seems unlikely that the striking features of the action of chloroform on their respiratory process will not be repeated in one way or other in higher animals.

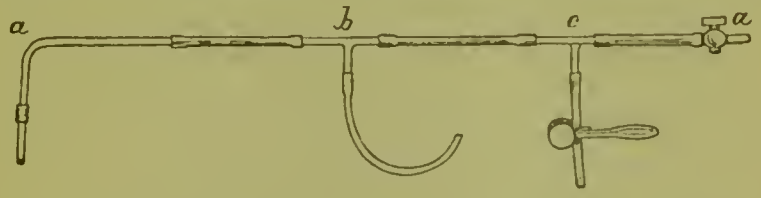
The series of experiments under discussion have been undertaken as a side issue in a research into the action of the uterus in rabbits, on which I have been engaged for some time. In my

[1] *British Medical Journal*, vol. ii., 1880.

experiments on this organ I found that very striking changes occurred in the respiratory rhythm as a result of stimuli applied to certain parts of the pelvic viscera and lumbar cords of rabbits; and it was in the course of the endeavour to determine the exact nature of these changes that my attention was arrested by the behaviour of the respiratory mechanism under the influence of chloroform.

As I could find in the literature of the subject no accurate description of the respiratory phenomena in anæsthesia, I had to proceed to its investigation. I shall have occasion to refer later on to the opinions scattered through the text-books and lectures on the subject, but meantime proceed to describe the mode of observation adopted.

Mode of Experiment.—The animal was fully anæsthetised by chloroform by means of a cone containing lint soaked in the fluid, and held over its nose. It was then secured to the holder, and tracheotomy performed, and a bent glass cannula tied in. The time required for this part of the operation varied with the greater or less susceptibility of the animal to the vapour. The variations were considerable, and from the time of first administration of the vapour to the completion of tracheotomy a period of from 5 to 15 minutes elapsed. The cannula *a* was connected by short pieces of



rubber tubing to two glass T tubes *b* and *c*. The branch of *b* was connected with a tambour or other recorder, and that of *c* was employed for administering the chloroform. This latter tube could be closed by a pinch-cock. The main tube ended in a vulcanite tap *d*.

By means of this vulcanite tap the effect of the air current on the tambour could be regulated: thus more or less extensive movements of the tambour lever could be obtained according as the tap was partially closed or opened. In the earlier observations a tambour writing on a continuous roll of paper by means of an ink-pen was employed. In the more recent ones, a slow moving smoked surface has been substituted; and instead of the tambour, I have employed a form of water-manometer devised by myself for such purposes.[1] This instrument possesses several advantages over Marey's tambour, which, I believe, render its indications more valuable and accurate. Its chief advantage is, that while it is as sensitive, if not more so, than the tambour for small

[1] This instrument will be described in an early number of the *Journal of Physiology*.

pressures, it is capable of recording accurately much greater variations than Marey's instrument can indicate. In the latter instrument the limit of elasticity of the rubber membrane is soon reached, when increments of pressure fail to effect proportionate movements of the lever. Thus the characteristic features of many tracings demanding such record are lost. The special nature of the tracings obtained by this instrument will appear further on.

I have explained that the chloroform was adminstered by means of the second T tube. The concentration of the vapour was regulated by the size of the receptacle for the chloroform and the diameter of its neck. The same size of tube was always employed; and if this were dipped into a narrow-necked bottle containing a large quantity of chloroform, the vapour would be in its most concentrated form, whereas were it placed in a large jar containing a small quantity of the anæsthetic on cotton wool, the vapour would be in a dilute condition. Further, the quantity given could be still further diluted by admitting a larger quantity of air by the tap, and by cutting off some of the vapour by partly closing the pinch-cock on the chloroform tube.

I made some experiments by having a Wolff's bottle attached to the main tube, and by connecting the registering apparatus to the other neck of the bottle; but as the results of several experiments did not vary from those of the simpler apparatus, I abandoned this arrangement as being needlessly cumbrous. From such an arrangement it will be seen that every inspiration will draw air out of the tambour, and will be indicated by a fall in the lever, while expiration will force air into the tambour, and will cause a rise of the lever. In the case of the records obtained by the manometer, the opposite is the case. The fall of the lever represents an expiration and the rise an inspiration. The apparatus having then been arranged as described, the animal was allowed to breathe air alone until signs of returning reflex appeared. The respiratory rate now generally fell to 7 or 10 respirations in 10 seconds—*i.e.*, from 40 to 60 per minute, varying chiefly according to the size of the particular animal. The clock was now started, and a tracing of the respiration taken for 10 or 15 seconds. The chloroform tube was then dipped into the receptacle, and the moment noted on the paper. In many of the tracings, the respiratory movement was recorded continuously until cessation. In others (these extending over several hours) the respiration was taken before and after each change in administration or withdrawal, and at regular intervals of time.

In this way I could note the effects of concentrated and dilute vapour, given continuously or intermittently, and I have therefore observed the mode of respiratory cessation under the following conditions:—

1. The continuous administration of the concentrated vapour.
2. The alternate administration of concentrated vapour and air.

3. The continuous administration of the dilute vapour.

4. The alternate administration of the dilute vapour and air. Further, as I have succeeded in resuscitating the same animal repeatedly after respiratory stoppage, I have noted the effect of the administration of chloroform.

5. After repeated cessation and re-establishment of respiratory movement.

6. After the withdrawal of blood from the vessels.

It is to be clearly understood that the observations I am about to describe deal with the respiratory phases under the influence of chloroform from the supervention of anæsthesia onwards, and have no bearing upon the changes during the period of the early action of chloroform up to the production of anæsthesia. The terms of my license do not permit me to make observations of this nature until the anæsthesia has been induced. The absence of an account of the early stages of chloroform action does not, however, I venture to think, rob the observations about to be described of practical interest, inasmuch as it is undoubtedly *after* the induction of anæsthesia that the grave danger from chloroform arises from oversight or misadventure.

I have already stated that the records of changes in the respiration by previous observers have been more or less indefinite and unsatisfactory, and that we can form from these statements no very distinct idea of the effects produced by the anæsthetic.

In the very elaborate report of the Chloroform Committee of the Medico-Chirurgical Society of London [1] I find the following under "The Effect of Chloroform on the Respiration:"—"The respiratory efforts were at first deep, but by degrees they became more and more shallow. With this loss of depth the respirations for a while retained their unnatural frequency, but after a time became less frequent than natural."

Sansom [2] says, "The breathing is quieted, but the rate of the respiration is scarcely altered, unless the narcotism is pushed to coma."

It would be difficult to reconcile these two statements.

Dr P. Knoll,[3] in an elaborate paper on "Chloroform and Ether," says, "The first change is a long expiratory tetanus, which may last 6 to 10, or even 16 seconds, and may be repeated several times. Respiration then remains a short time greatly retarded, often partly, sometimes entirely, owing to the prolongation of the respiratory phase, then follows the final acceleration, with still deep inspiration, but becoming gradually shallower, until it entirely ceases."

Wood [4] says, "The respiration may be at first rendered slower by chloroform, but after a time are generally quickened, and as

[1] *Trans. Medico-Chirurgical Society*, vol. xlvii., p. 323.
[2] *On Chloroform*, p. 63.
[3] *Sitzungb. Wiener Akad.*, 1876.
[4] *Treatise on Therapeutics*, p. 288.

inhalation is persisted in, they become more and more shallow, irregular, and distant, and finally cease."

Several other authorities may be cited, but these will serve to show that the general impression is, that after certain phases admitted by some and denied by others, the respiration becomes slower, shallower, and finally ceases. As to how far the experiments about to be described correspond with any of those statements will appear later on. It would serve no good purpose to occupy the time of the Society by a detailed description of the individual experiments. I purpose rather to give a general account of the results arrived at, and will only refer to the details of such experiments as may be necessary to establish my conclusions. I shall therefore consider—

I. *The Effect of the Continuous Inhalation of Concentrated Vapour, from the Induction of Anæsthesia until Cessation of Respiration.*

A well-grown rabbit is fully anæsthetised, secured and connected with the recording apparatus as described. Air only is breathed, until the conjunctiva reflex appears, and the respiration has fallen to 6 to 10 in 10 seconds. If the chloroform tube is now dipped into the narrow-necked bottle, containing as much chloroform as will bring it to within half an inch of the tube, the tambour will indicate the following changes :—Soon after the tube is dipped in, a noticeable change occurs in the respiratory rate. The first 2, 5, or 10 respirations may show no signs of change, but at the end of this interval the rate begins to rise. Coincident with this increase in rate we almost invariably find a marked increase of volume. This increase does not take place suddenly, but each succeeding respiration is slightly deeper than its predecessor, and soon the maximum depth is attained. This maximum condition is maintained for a very short period, sometimes for not more than 2 or 3 seconds, and then the volume begins gradually and regularly to diminish. Moreover, at the moment the respiration has attained its maximum volume it has also assumed a definite rate, which is maintained up to the very close of the respiratory movements with almost absolute uniformity.

In a typical case of respiratory cessation, due to the continued action of concentrated vapour, we have the following stages (the animal being fully anæsthetised) :—

1. A latent period of 3–5 respirations.
2. A period of quickening rate, and increasing volume.
3. A period of constant rate, and diminishing volume.

The earlier observations being made on continuous paper, the records themselves are too long to be conveniently handed round, and the waves are too far apart (the paper moving rather rapidly) to show the gradations of the phases satisfactorily. But the

special features in the change of volume will be shown in the tracings which I hand round, and the variation in rate from administration to cessation will appear in the following table:—

TABLE showing the Respiratory Rate in Successive Periods of 10 Seconds from Administration of Final Dose till Cessation (Concentrated Vapour).

		I.	II.	III.	IV.
Sex of animal,		F., ¾ grown.	F., full grown.	M., full grown.	F., full grown.
Time from commencement of chloroform,		15 min.	25 min.	18 min.	30 min.
Respirations in 10 seconds before lethal dose,		10	12	10	7
Respirations in successive periods of 10 seconds during administration of lethal dose,	0–10	12	18	13	10½
	10–20	17	18	14	11
	20–30	18	20	14	11
	30–40	23	20	15	10
	40–50	26	20	14	12½
	50–60	26	20	15	12
	60–70	26	20	14	12
	70–80	25	20	14	10
	80–90	25	19	14	12
	90–100	...	20	14	12
	100–110	...	...	14	12
	110–120	...	...	14	
	120–130	...	...	14	

It seems to me unnecessary to detail more examples of this nature in this fashion. Many more could be given, but they all bring out the same feature, namely, that after the maximum rate is established (what I may call the cessation rate), this rate continues, with scarcely any alteration, until the very end.

It is not possible to give, except by a tedious and elaborate system of measurement, a description of the changes in *volume* coincident with these changes in rate. But a glance at the tracings taken from the original records which I show, will bring out what I mean to indicate. Take, for instance, the tracing Figs. 1 and 2, Plate I., which is the first I obtained, and which was the first to call my attention to the curious uniformity of the rate. The animal, a full grown doe, had been breathing ether for some hours, and in order to kill it I substitued chloroform, with the result shown in the tracing, namely, a gradually diminishing volume, with an almost absolutely constant rate.

Again, the tracing Figs. 3, 4, and 5, Plate I., obtained from a full grown buck, shows the same features as I have described. The first line, Fig. 3, shows the rate previous to the administration of the fatal dose of chloroform, the rate being 5½ in 10 seconds. The second line, Fig. 4, shows the rate and volume 10 seconds after chloroform was begun. At the beginning of the line the volume is deeper than in the first line, and the

rate 19 per 10 seconds. Towards the end the volume is diminishing, and the rate in the last 10 seconds of that line is 23. Line 3, Fig. 5, 30 seconds after Fig. 4, shows the cessation of the respiration, the volume gradually diminishing, but the rate remaining constant at 22 or 23 per 10 seconds.

But the special features of this form of respiratory death become much more distinct when we turn to the tracings obtained by means of the slow moving drum. In these the whole of the changes are brought together under the eye at one moment.

I first show two tracings, Figs. 40 and 41, Plate IV., taken from the same animal, it having been resuscitated after the first one. The rabbit was a full grown male, which had been 18 minutes under chloroform. After tracheotomy, the respirations had sunk to 12 per 10 seconds, Fig. 40. Chloroform was given at the point marked (*Ch*), and it will be noted that the respiration, which has been somewhat irregular previously, becomes steady, and of from 12 to 14 crests. The volume then sinks slightly, and immediately after begins to increase, the rate being somewhat accelerated. Soon it reaches its greatest volume, then gradually diminishes, the rate being maintained up to the very end. Just before ceasing, the slightest possible increase in volume appears, but lasts a very few seconds, and the end of the tracing shows a gradual diminution and final disappearance of the curves.

A similar description applies to the tracing Fig. 41 which follows. The animal on the conclusion of Fig. 40 was quickly resuscitated, and when the breathing reached 10 to 12 in 10 seconds, chloroform was again given, with a similar result. In this case the deepening at the end is even more marked than in the previous one, and the other features are almost identical.

The tracing Fig. 42 was obtained from a three-parts grown female, and does not require more detailed description. The tracing is much shorter than the previous one, but the feature of diminishing volume and constant rate are clearly brought out.

The tracings Figs. 43 and 44 are similar in main features to those already described, but show some characters which I shall have to refer to shortly. Fig. 43 was obtained from a large male rabbit which had been 40 minutes under chloroform before the tracing was commenced. Air only was breathed up to part marked "air." Chloroform being given, the respiration is unaltered for two or three breaths, then it gradually rises in rate and volume, then the volume diminishes, the rate remaining the same. Unfortunately the drum ran out before the cessation occurred, and the last part of the tracing is not given, but one can safely fill up the gap from the experience of other cases. The special features of this and the tracing Fig. 44 I must leave for subsequent notice.

The period required to produce death by chloroform given in this manner varies with many circumstances, notably with the size of the animal; but it seems a most interesting point that, after

the respiration rate is fairly established coincident with the maximum volume being attained, we should have an absolute maintenance of that rate down to the very last gasp.

So far as I am aware, this feature of chloroform death is not recorded in the description of any of the numerous experiments which have been made on this anæsthetic. The gradually increasing feebleness of the respiratory movements has been noted by all observers. The diminution of the heaving of the chest-wall is obvious to the most casual onlooker, and it was perhaps natural to assume, in the absence of definite demonstration to the contrary, that the reduced volume was accompanied by a diminishing rate. Such, however, is not the case, so far as a very considerable number of observations seem to show under the condition I have discussed, namely, continuous administration of concentrated vapour.

So far, then, we are justified in concluding that in rabbits a fatal dose of chloroform vapour given under anæsthesia affects the respiratory centre in two stages.

1. A short stage, in which the explosions of the respiratory centre are quickened in rate and increased in intensity—a stage of "acceleration and augmentation."
2. A longer stage, in which the explosions occur at the previous quick rate, but in which their intensity becomes gradually less and less, until they disappear.

While considering the action of such concentrated doses, there are one or two points which demand attention.

1*st*, When we compare more minutely the character of the breathing in the two stages just referred to, we shall find certain characteristic features which are worthy of notice. I have said that there is a *gradual* increase of the extent of movements in the earlier stage, and a gradual diminution in the second. So far as the majority of the earlier observations show, this diminution in the second stage would seem to be perfectly regular, *i.e.*, each inspiration is somewhat less deep than its immediate predecessor. These earlier observations were made by means of tambours furnished with writing points, and were taken on continuous paper at a comparatively rapid rate, and under a very low air pressure. In some cases, when the air pressure was considerable, the tambour indicated a remarkable irregularity in the second stage, in which, while the rate remained constant, every third or fourth breath seemed deeper than the others. This irregular rhythm is well shown in a tracing, Fig. 12, Plate I., taken from an animal which had been breathing ether for a time, and in which death was being induced by chloroform. It is further seen in Fig. 13, Plate I., taken from an animal also dying under concentrated chloroform, and in which the left vagus had been cut.

But the characteristic features of this respiratory rhythm come out in a record obtained by means of the water manometer to which I have referred. This apparatus possesses the advantage

over the tambour that its indications are absolutely quantitative, so that we have the value of each respiration accurately recorded. Further, it gives very delicate indications of low pressure, and can thus be employed while interfering as little as possible with the normal respiratory currents. On turning to a tracing obtained by this instrument, Fig. 43, Plate IV., it will be seen that so long as air is being breathed the volume of each respiration is identical—that when chloroform is given the movements gradually and regularly increase in volume, until a maximum is reached, but that when succeeding this the movements begin to diminish in volume, this diminution is not a simple gradation, but consists of a succession of waves of increase and diminution, though the total volume is undergoing absolute decrease. The result is that we have a succession of phases, each of which may reasonably be compared to a Cheyne-Stokes' phenomenon, consisting, as it does, of a succession of waves leading up to a maximum, followed by another succession leading down to a minimum. The existence of these phases of the second stage are also beautifully shown in the tracing Fig. 44, Plate IV., obtained under similar conditions to the others.

It seems somewhat difficult to account for this remarkable rhythmic variation, unless it be that these larger waves represent the normal discharges of the respiratory centre appearing coincidently with the chloroform acceleration, and thus asserting themselves in this modification of the narcosis respiration.

2*nd*, During early anæsthesia, while consciousness is entirely abolished, the action of the vagus is distinct and easily demonstrated. Stimulation of either the trunk or the divided central end results in inhibition or quickening of the respiration, according to circumstances. Further, during the first stage of increased volume rate (the result of a lethal dose) this activity remains, though in a gradually diminishing degree; but on the accession of the second stage its action is abolished, and when the stage is fairly developed the strongest currents fail to produce any response through this nerve. This is well shown in Figs. 9–11, Plate I., and Fig. 45, Plate V. In the first of these tracings, Figs. 9–11, Plate I., the animal, after being anæsthetized, was allowed to breath pure air for some time. A weak current (20) was thrown into the left vagus, and almost complete respiratory cessation followed (Fig. 9). Chloroform was given at the point *Ch.*, Fig. 10, and still the same effect follows the same current a few records afterwards. Fifteen seconds elapse between 10 and 11, and now it is seen that little or no effect follows the introduction of the same strength of current, and even very strong currents seem, a little later on, to produce but the faintest impression. Fig. 45, Plate V. shows a similar result. The explanation of this seems to be that in the early stage the excito-respiratory effect may be due to the action of chloroform on the pulmonary terminations of the pneumogastric, during which period these

stimuli are carried to a centre capable of being affected by them, while in the later stage, the phenomena of which are due to the paralysis of the centre, afferent currents, however strong, fail to produce any effect whatever.

3rd, A point of some practical interest deserves notice. How will the final result be affected by the withdrawal of the vapour during any of these stages? How far may the chloroform be continued with safety? Numerous observations have shown me that the withdrawal of the vapour during the early stage (*i.e.*, before the respirations have reached their maximum) is usually followed by recovery of the normal rhythm after a time, but, on the other hand, the continuance of the administration into the second stage, and its withdrawal after diminishing volume appears, is not followed by such recovery, but that the gradually diminishing volume continues, until it is finally extinguished.

II. *Action of Concentrated Vapour of Chloroform given Intermittently.*

If an animal be kept under the influence of the vapour for a lengthened period, by its frequent administration and withdrawal, we find that in the early part of the experiment the characteristic quickening of the respiration will follow each dose, and that the return to the normal rhythm will gradually succeed its withdrawal. Later on, however, certain remarkable phenomena appear, with sufficient frequency to warrant us in attributing them to the special way in which the vapour is given.

1. Death may occur in one of several ways.
   - *a.* In some few cases where the experiment has lasted less than two hours, the mode of death is similar to that described under the previous heading.
   - *b.* In a much larger number of cases the respiration ceases with a marked slowing and irregularity of the waves. This is well shown in the case of an animal which had been under the influence of intermittent doses of the concentrated vapour for upwards of three hours, and in which the cessation rate produced by the final dose was in successive periods of 10 seconds—0–10, 21; 10–20, 21; 20–30, 21; 30–40, 20; 40–50, 20; 50–60, 19; 60–70, 17; 80–90, 15; and the final curves are exceedingly irregular, as seen in Fig. 22, Plate II.
   - *c.* In the third place, death may occur during one of the administrations with remarkable suddenness. I have frequently observed that after an animal had been breathing air for a period of, say, ten minutes, and when the vapour was being reapplied, heaving movements of the chest occurred and then suddenly ceased, the respirations immediately preceding such cessation being quite

deep and full. This form of cessation is well seen in Fig. 46, Plate V.

2. Besides these variations in the mode of death, I have observed a remarkable condition exhibited by rabbits under the intermittent concentrated vapour, which I venture to think worthy of notice, from a practical as well as theoretical point of view. I do not venture to speak with entire confidence as to the explanation of the condition, as I have not been able to reproduce it sufficiently often to enable me to arrive at a definite conclusion regarding it. As the practical bearing of the point it raises is considerable, I have thought I might be justified in bringing it under notice, though as yet inadequately investigated, in the hope that it may receive some attention from those in circumstances to study it.

It frequently occurs that when a rabbit has been kept for a considerable time under the intermittent administration of chloroform the respiratory volume changes with great suddenness, and from being deep and regular becomes shallow and wavy. Examples of this will be seen in Figs. 16 and 25, Plate II. On the other hand, the converse may occur, and the volume suddenly increase. Moreover, these changes occur without any apparent increase or variation in the extent of the chest and abdominal movements, so that a light lever laid on these regions does not show any sign of the alterations referred to.

But further, I have now tracings from six cases, in which, with little or no previous alterations in rate or rhythm, the air current, as indicated by the recorder, comes to a sudden standstill, the pen drawing a straight line. But while this was so, the movements of the chest still continued, neither their rate nor their extent being appreciably altered. Such a state of matters may last for an uncertain period, but is usually ended by the air current recommencing in the tube. These tracings are reproduced in Figs. 14 to 21, Plate II.

It will be observed that the line indicating absence of the air current continues for a considerable length of time,—16, 44, 21, 32, and 28 seconds in five cases recorded.

It is further to be observed that the respirations immediately before and immediately after the cessation of the air current are not specially remarkable. In some they are slightly exaggerated, in others slightly irregular. In all those referred to, the animal had been some hours under chloroform. In the sixth example of this nature, Figs. 19 to 21, Plate II., the tracing is altogether remarkable. The animal, a full grown male, had been under chloroform for $2\frac{3}{4}$ hours, and respiration had ceased under its influence. Resuscitation was effected by artificial respiration, and immediately thereafter the recorder was connected and the tracing Fig. 19 taken. It will be noted that the line traced by the pen shows distinct though shallow respiratory waves, indicating a certain air current; at *a* the tube was momentarily pinched, with the result of producing a

sudden crest of considerable height. This is followed by six respirations of ordinary depth, then it sinks again to the previous shallow state. At *b* the tube was again pinched with a similar result, and again at *c*—this time only a deeper wave being produced. 13 seconds after this, however (at *d*), 7 deep respirations occur spontaneously, followed by a considerable number of average respirations. Again the shallow type supervenes, and then again is followed by a succession of greatly exaggerated waves. Now, during all these various changes in the recorded movements it is to be noted that the rate is absolutely the same, 9½ crests in 10 seconds. And further, it was observed at the time that the respiratory movements in the chest and abdomen were as distinct during the occurrence of the shallow breathing as during those showing normal volume, and that these movements were but slightly increased during the very deep waves which appear from time to time.

Now, this cessation of the air current, seen as occurring totally in the first five observations referred to, and its modification in this remarkable way in the last one described, can only be accounted for, so far as I can see, in one of two ways.

1. Either a plug of some sort formed in some portion of the apparatus so as to cut off the air from the recorder; or,

2. It must be accounted for by some alteration in the action of the breathing apparatus, by which, while still continuing to move, it failed to effect the regular inflation and compression of the lungs, so that the air column came to a standstill—that, in fact, while the thoracic walls seemed to be performing their natural movements, no air was passing out and in.

Now, I believe I am justified in excluding the plug as an explanation of these tracings on the following grounds:—

1. Dipping the chloroform tube under the level of the liquid was followed by merely an almost imperceptible rise of the fluid in the tube. This showed that the plug was not simply in the tambour tube, there being no current in the main tube.

2. The tracheal tube was made of glass, and any plug or obstruction there could be seen.

3. Had the air current been cut off by a plug while the normal movements continued, it is obvious that the latter would have been much exaggerated from the dyspnœa induced.

4. The mechanical closure of the air tube for even a few seconds in an animal breathing in the ordinary way is always followed by violent thoracic movements which at once attract attention.

It has long been recognised by surgeons that thoracic and abdominal movements are no proof that air is entering the chest during anæsthesia, and an administrator of chloroform always assures himself of this fact by actually feeling or hearing the air current. The stoppage of the air current with continuation of

movements of the abdominal and thoracic walls is usually explained in the human subject by the tongue falling back and the glottis becoming closed. But in these animals the air enters by the cannula below the glottis, and this explanation cannot hold good here.

I can only explain the cessation of the air current just described by attributing it to a failure in the co-ordination of the muscles of expiration and inspiration, and that, in fact, a respiratory stammer is induced. Thus it might come about, through the action of the anæsthetic on the centre of respiration, that the action of the muscles of inspiration might be antagonised by the action of the muscles of expiration, and *vice versa*. Further, this inco-ordination might equally apply to the muscles of forced respiration were these called into play. Thus rhythmic movement of the chest might continue to the eye, and, even though a tendency to dyspnœa was induced, it would find no expression in the disordered mechanism.

It seems to me no great straining of our experience to suppose that a powerful drug like chloroform, circulating in the blood for a lengthened time, might be quite capable of disturbing the rhythmic action of the centres of expiration and inspiration. We are familiar with the action of alcohol on an associated centre, that of speech, and I see no reason to doubt that a comparable effect may be produced by chloroform on the respiratory centre.

If this is so, it bears on an important practical point, and seems to accentuate the importance of disregarding abdominal and costal movements as an indication of the continuation of respiratory currents during anæsthesia, and of the administrator making certain, by actually feeling it, that air is actually passing in and out of the lungs. Another observation in connexion with this is worthy of note. In an experiment, in which the animal had been under chloroform for several hours, the paper web ran out, and, while it was being readjusted, the tambour ceased working while the movements of the chest continued. On pinching the eyelid with a pair of pointed forceps the movements of the tambour at once recommenced. Some time afterwards, the tambour again stopped, and again began to move when the eyelid was pinched. It seemed natural to suppose that the shock of the afferent impulse in some way or other served to harmonize the disturbed action of the centre of respiration, and it might be worth considering in this connexion how far the resumption of natural respiration, after the dragging forward of the tongue in certain cases, may not be the result of a similar reflex, and not merely a removal of an obstruction to the entrance of the air. At the same time, I do not press the theory I have advanced. I have as yet had no satisfactory opportunity of determining all the elements of the condition. I have brought the point under the notice of the Society for the purpose of drawing attention to it, and, if possible, inducing any one who may have the opportunity to give it some consideration. I feel certain that the phenomena are not due to a plug, and it seems essential to assume the occurrence

of some remarkable modification of the respiratory mechanism in order to explain them.

III. *The Continuous Action of the Dilute Vapour.*

The method I adopted of administering the dilute vapour was to put a certain amount of chloroform on a piece of lint into a large wide-mouthed bottle and place the tube in the neck. In this way the vapour was largely mixed with air.

If the administration was continued from the induction of anæsthesia until cessation occurred, the difference between the action of the strong and dilute vapour lay chiefly in the length of time required to stop respiration. Frequently it would seem as if the final diminution of the volume had set in, when quite suddenly the volume would increase and continue unaffected for a long period. Finally, however, the cessation occurred, and presented the same character of constant rate and diminishing volume which have been described.

IV. *Action of the Dilute Vapour given Intermittently.*

Characteristic phenomena usually accompany this mode of administering the dilute vapour.

It is possible to keep a well-grown rabbit in a complete state of anæsthesia for many hours by the careful administration of dilute chloroform from time to time. If this condition is kept up for $1\frac{1}{2}$ hours or longer, and chloroform given and continued until respiration finally ceases, it will be found that almost invariably death comes on accompanied by gradually diminishing rate and volume. The respirations become more and more shallow and more and more distant.

This is shown strikingly in the records of two observations on Plate III. In the first (Figs. 26 and 27), the animal was a full-grown female, which had been under the anæsthetic for $2\frac{3}{4}$ hours. The record is a double one—A and C, which are continuous, being taken by a tube in the œsophagus, and B and D, also continuous, by a stethograph. The remarkable slowing is well shown.

The second record, Figs. 28 to 32, was obtained from a three-parts grown male under chloroform for 3 hours. The breathing, at first extremely irregular, becomes quieted under the action of the final dose, and shows in a remarkable fashion the peculiar mode of death.

So far, then, I conclude that in rabbits slowing of the respiration towards death occurs when they have been kept for a lengthened period under the dilute vapour, and this prolonged condition can only be obtained by intermittent administration.

V. *Effect of Loss of Blood on the Respiration under Chloroform.*

In some of my earlier observations I found that the acci-

dental wounding of a vessel in the neck during a dissection, if followed by loss of much blood, was almost invariably succeeded in the course of 5 to 10 minutes by a slowing of the respiration, and by a peculiar jerky movement of the diaphragm observed by its effects on the viscera when the abdomen was opened. This occurred so distinctly in several cases that I took a tracing of the respiration in the ordinary way in the case of a full-grown rabbit, whose jugular has been opened, and which had lost a good deal of blood. The tracing, as shown in Figs. 23–25, Plate II., is sufficiently characteristic to be worthy of notice. Fig. 23 shows the effect 15 seconds after the blood was withdrawn; Fig. 24, 1½ minutes thereafter; and Fig. 25, 5 minutes after Fig. 24. The first line shows the commencement of a peculiar wavy respiration. This is, however, seen to a very much more remarkable extent in the second line, where the breathing is much slower, and in which the notches of the curves are repeated with great exactness. In the third line the rate is again rising, the notched character of the waves still being present. At a certain point the volume suddenly falls, the rate remaining much the same. This curious alteration in the nature of the curves is interesting in connexion with another tracing, to which I shall direct attention immediately. Whether it has any practical bearing on the effect of hæmorrhage during operations I cannot venture to say. In rabbits it certainly is an almost constant sequence of anything like serious loss of blood. It would be of much interest to know whether any such alteration of the respiration has been observed by surgeons in operations involving the loss of much blood.

It seems to suggest the question as to how far the respiratory centre depends for its rhythmic action on a certain *vascular* tension in the brain. May it not be that the diminution of the tension following the loss of blood results in an interference with the rhythmic discharges from the centre, and hence the secondary waves seen in these curves?

As will be seen, the records give no account of the pulse rate or blood pressure during the respiratory changes.

The points of chief interest in such a relation are:—1. The changes in the blood pressure during the narcosis; 2. The relative moment of cessation of cardiac action and respiration.

The first point has been investigated with the greatest fulness (as I have already said) by both committees referred to, by Knoll,[1] Kratschmer,[2] and others, and nothing further seems to be done in that direction.

With reference to the second matter, the relative moment of cessation of the two movements, opinions do not quite coincide, but all are agreed that the stoppage of the heart before the cessation of respiration is a comparatively rare event in animals. The Committee of the Medico-Chirurgical Society[3] determined the moment of

[1] *Loc. cit.* [2] *Sitzungb. Wiener Akad.*, Oct. 1870. [3] *Loc. cit.*

cardiac stoppage sometimes by means of the movements of the kymograph, and in other cases by means of the movements of a needle pushed into the tissue of the heart through the thoracic wall.

They mention four cases in the report in which the heart stopped first, and this happened out of many experiments. The Glasgow Committee[1] had only one animal whose heart stopped before the respiration, and this was out of nearly a hundred experiments.

In none of my experiments on nearly fifty animals has the pulsation in the carotid stopped before the respiration. Usually it continued 1 to 2 minutes after, in some cases as long as 4 minutes.

In my earlier experiments I determined the moment of stoppage of the pulse by holding the vessel between the thumb and forefinger, but in my later observations I have employed an instrument which I have devised for recording the pulsation in unopened and unligatured vessels, which records have not, as far as I know, been previously obtained.

By this instrument, which I have termed the "Arteriograph," we are able to detect and record the faintest pulsation in the artery, without the trouble or source of error arising from the manometer.[2] The cutting off of part of the blood supply to the brain, following ligature of the carotid previous to connexion of the manometer, must be inevitably a source of fallacy with regard to investigations into the action of substances such as chloroform, etc., and I should venture to hope that in such investigations, where *pulse rate* is required, the instrument will prove of some use.

In all these cases in which I have employed it I have found that the cardiac movements continued a considerable time after the respiration had ceased—in the case of strong doses, beating vigorously; in the case of dilute doses, beating more feebly.

No doubt there are cases in which the heart gives in first, but they are very rare, and do not affect the results I have arrived at as to breathing, as all the various phases of cessation have occurred with the heart in action.

To summarize then, briefly, the results obtained by these experiments, I find that in rabbits already anæsthetised by chloroform respiration ceases, according to the mode of administration, in the following ways:—

A. The continued administration of the concentrated vapour.

1. Respiration shows three periods—
   - *a.* A latent period of 2-10 unaltered respirations.
   - *b.* A period of augmented volume and accelerated rate.
   - *c.* A period of diminishing volume and constant rate.

[1] *Loc. cit.*

[2] I must leave the description of this instrument and some of the results obtained by its means for another paper.

2. This last stage shows two sets of curves, the larger possibly due to the normal discharges of the centre added on to the more rapid discharges caused by the chloroform.
3. During this stage the action of the vagus becomes gradually abolished.
4. Removal of the vapour during the stage of acceleration is usually followed by recovery, but in the last stage such removal does not affect the fatal issue.

B. The intermittent administration of the concentrated vapour. Respiration may cease in three different ways :—
1. In experiments under 2 hours the cessation occurs with diminishing volume and constant rate.
2. In others, the respiration slows somewhat towards the end, and shows great irregularity.
3. The respiration may come to an abrupt cessation, generally when the vapour has been given after air has been breathed for a long interval.
4. During the prolonged administration the air current may suddenly come to a standstill while the respiratory movements continue.

C. The continued administration of dilute vapour acts in a similar way to the strong, only the time occupied is longer.

D. The intermittent administration of the dilute vapour is followed by great slowing of the respiration towards the end.

E. Loss of blood produces an irregular " wavy " respiration.

F. The heart is affected after the respiration, and is less affected by concentrated doses administered continuously than by dilute doses given intermittently.

Restoration of Breathing after Respiratory Cessation.

Were death by chloroform much rarer than it unfortunately is, the study of resuscitation of subjects succumbing during its administration must be always one of deep interest.

The subject has been discussed at length in many papers, and fully treated in all works on anæsthetics, and I have no intention of entering on any consideration of the various methods which have been recommended or suggested to effect restoration of the breathing in those in which it has ceased.

Experiments on animals and accumulated experience on the human subject have gone to show that of all the means which have been recommended with this object artificial respiration is the most efficient. The importance of immediate resort to the method has been insisted on by Snow, Sansom, Anstie, Richardson, West, etc., the Committee of the Medico-Chirurgical Society, the Glasgow Committee, etc.

I need scarcely say that my own observations tend entirely in the same direction.

The method of carrying out such artificial respiration is usually one of three :—

1. Marshall Hall's, or Sylvester's.
2. Tracheotomy, and insufflation of lungs through the opening.
3. In simple cases, especially children, by mere compression of the chest, allowing it to fill by its own resiliency.

There is no doubt that a very considerable number of lives have been saved by the timeous adoption of one or other of these methods.

Unfortunately, however, cases occur from time to time in which the patient employment of one or all of these methods fails, and the cessation of respiration becomes permanent.

In the series of experiments on the uterus, to which I have alluded, and in which I was led to use chloroform as the anæsthetic instead of ether, from the efficiency of the former drug in subduing peristalsis of the uterine horn, my observations were frequently interrupted by the respiration ceasing owing to inadvertence in administering too much chloroform. Accordingly, I had frequently to practise artificial respiration with a view of resuscitating the animals. This was effected by simply blowing into the tracheal tube at intervals, and so distending the lung and allowing the air to be expelled by its collapse.

Such a process I found successful in perhaps two animals out of three. But if the same accident occurred twice in the same animal, resuscitation was, if successful a second time, effected only with great difficulty, and in most cases it entirely failed.

While performing the insufflation in the case of some of these animals I was struck by the large quantity of the vapour which smell and taste showed was present in the air expelled from the chest at each recoil of the lungs. It was obvious that the saturation of the air in the lung cells with chloroform must be a serious bar to the re-establishment of respiration, and that the first indication was, therefore, to endeavour to get rid of this vapour as quickly as possible.

For, consider for a moment the sequence of events when we forcibly inflate the lungs of an animal which has stopped breathing under chloroform. As I have already stated, experience has shown that the heart almost invariably continues to act after respiration has come to a standstill. Moreover, in those cases in which the heart has already stopped it recommences its beat before the first resumption of spontaneous respiratory movements. Accordingly, the increase of tension in the lung following the first forcible inflation must inevitably result in the diffusion of another dose of the fatal vapour into the blood, which sweeps it on to the centre, and there extinguishes any beginnings of renewed action.

I venture to think that nothing more than this is needed to

account for the "few spasmodic" gasps which we repeatedly read above followed attempts at reanimation by Sylvester or Hall's method, in the account of fatal cases of chloroform poisoning. The statistics of the Medico-Chirurgical Society's Committee contains the record of many such cases. In such cases it seems to me likely that the movements of the lungs caused by the commencement of artificial respiration, mechanically or otherwise, excites the heart to somewhat more vigorous action. The respiratory centre is excited by the increased blood pressure, and a few discharges occur, but its activity is soon extinguished by the stream of blood now coming from the lungs laden with the deadly vapour, which has been forced into it in the way I have indicated. Accordingly, acting on this idea, in my next experiment I commenced attempts at resuscitation by sucking air out of the lung through the tube, and simply allowing the lung to fill with air by its own recoil. This process was aided by compressing the chest wall gently while sucking the air out. In the course of a few seconds the nostrils began to move, and after five or six such aspirations the natural breathing reappeared and soon passed into gentle respiration. After a little time the respiration was again stopped by means of chloroform, and again resuscitation was effected by a similar method—this time requiring a little longer for its accomplishment. The same process was repeated twice again, four times in all, on this animal—the last commenced 25 seconds after breathing had ceased, and occupied about 1½ minutes before breathing was fairly established.

Since that experiment I have made a large number of observations on this method, and certainly have found it most efficient. In only one case have I found it fail in adult rabbits, resuscitation being readily effected four, five, or six times, even after the animal has been upwards of two hours under the influence of the vapour, and in deep anæsthesia all the time.

The successful repetition of the experiment oftener than four or five times was, however, usually prevented by the air passages getting obstructed by a pink frothy exudation which developed in them, and which I attributed to the strain in the bloodvessels caused by the frequent exhaustion of the air cells. I never saw this appear until after the third or fourth time.

The desire to avoid this, however, caused me to hit upon the following modification, which I have employed recently and with greater success than simple inflation. This process, which I may term "perflation," I carry out in the following way:—Disconnecting the rubber tubing I take the end of the T tube attached to the tracheal cannula in my mouth, and closing the branch with the finger, I make one or two aspirations of the lungs, compressing the chest gently at the same time. This removes a considerable quantity of vapour from the upper passages. Then opening the branch I make a series of deep inspirations. The air rushes in by

the branch, and no doubt the greater part passes into the mouth. Yet some of it enters the lung, and a current is thus established by which a very large quantity of the chloroform is rapidly expelled, as can be proved by the taste of the air coming through the tube. After two or three such inspirations the taste of the vapour becomes fainter, and as soon as this is noticed I reverse the process, now blowing air into the tube, with force just sufficient to cause the chest wall to move in the slightest possible degree—the branch tube being open all the time. Generally, after one or two such perflations the heart shows signs of vigorous action, and shortly thereafter breathing commences and continues in a perfectly natural manner. Should it not return so rapidly, and after I am assured by the absence of taste or smell in the expired air that the chloroform has been almost entirely removed, then I close the branch tube and commence gentle inflation of the lung in the ordinary way.

By such a method I have resuscitated animals as often as ten times after cessation of respiration, and in these I have little or no indication of the production of the pink froth to which I referred.

Of course, in order to accomplish resuscitation so frequently as eight or ten times it is necessary in the later stages to lose no time between cessation of movement and commencement of the effort, but the mere fact that it can be effected under any conditions so frequently speaks strongly, I think, for the efficacy of the method. In the earlier resuscitations we may safely wait 30 to 60 seconds before beginning the attempt, provided the animal has not been for a long period under the influence of the vapour. The time occupied in the operation is much less when we begin before the heart has ceased to beat, but I have repeatedly waited until all indications of the heart's action had ceased, both as indicated by the apex beat and the pulsation in the carotid. But in such cases it has been an invariable experience that the heart resumed beating a considerable time before breathing commenced.

Speaking broadly, as regards the difficulty of resuscitation as indicated by the time required to effect it, I have observed that *the time required to restore respiration varies inversely as the concentration of the dose, and directly as the time required to stop respiration.* That is to say, the more concentrated the dose the easier was the reanimation, and the longer respiration continued under the action of the vapour the more difficult was the reanimation. This observation corresponds with that made on the same point by the Committee of the Medico-Chirurgical Society.

Thus the greatest difficulty was experienced in reanimating animals which had succumbed under the long action of dilute vapour intermittently administered, and in which respiration ceased with great slowing of the rate, as in Figs. 26–32. This

appears to me to be explained by the fact that chloroform is primarily a respiratory poison, and that, if given in sufficient quantity, it stops the action of the respiratory centre before it affects the heart. Accordingly, the heart may be beating vigorously some time after the respiration has ceased under the concentrated vapour. On the other hand, the prolonged circulation of the vapour in the blood ultimately affects the cardiac action, and, consequently, under dilute chloroform, the heart may stop with or immediately after the respiration, and hence the difficulty of resuscitation.

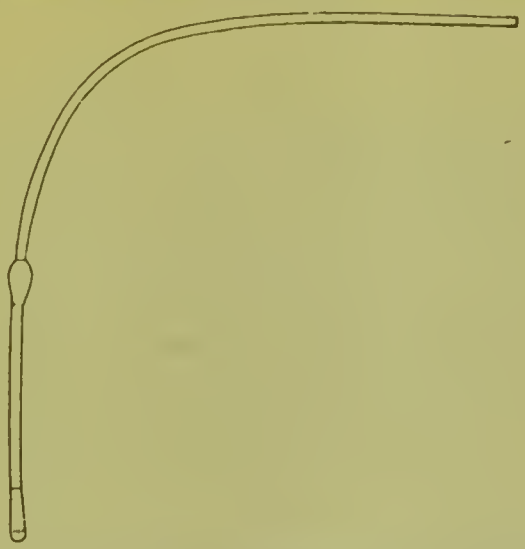
To show how well the characters of respiratory cessation under concentrated vapour which I have described are retained, even after repeated reanimation, I show tracings of the final stages of four cessations in the same animal, Figs. 5, 6, 7, 8, Plate I.; where it will be seen that it is only in the last one, when the animal had succumbed four times, and been more or less under the vapour for 4½ hours, that any slowing is observed, and even then it is not great. So also in Fig. 47, Plate V., taken after seven resuscitations, we have a similar correspondence with the mode of death occurring under the earlier administrations.

The observations recorded in this communication touch more than once on points of practical interest, but which time will not permit me to allude to. I content myself at present with a reference to one matter only, namely, that of resuscitation after respiratory cessation.

It is with diffidence that I venture to offer any suggestion whatever on so grave a matter as the method of re-establishing respiration in such cases, and this all the more, when the suggestion refers to a mode somewhat different from that ordinarily employed and sanctioned by experience. But the fact remains that the ordinary method sometimes fails in its object, which failure may, in certain cases at least, be accounted for by a defect which I have indicated. Encouraged, then, by the success of the experiments on animals peculiarly susceptible to the action of chloroform, which has followed the method I have described, and convinced of the physiological soundness of the principles involved in it, I would venture to suggest that in any case of respiratory cessation under chloroform (with the heart beating after the respiration had ceased), and where the removal of the vapour from the face, the drawing forward of the chin, and compression of the chest have failed to restore breathing, that the method of "perflation" should be tried without delay.

It might be done in lieu of a better means by a gum elastic tracheal tube as figured, provided with a conical collar to make it fit tightly into the glottis and prevent it slipping too far in. This could be slipped over the tongue and into the glottis, and the collar pressed down into the rima. The mouth is applied to the tube and several deep inspirations made. The mouth is with-

drawn from the tube after each inspiration, in order to let the lungs of the patient fill with pure air by the recoil. When this has been



repeated several times, the tube might then be slightly withdrawn so as to separate the collar from the glottis, and the process of perflation commenced by making deep and forcible inspirations. Air will rush into the trachea by the side of the tube, a current will be established, which will tend to carry air into the lung and sweep out the vapour. So long as any distinct odour of chloroform accompanies the air withdrawn the process should be continued, provided the natural respiration does not return. When all indications of the presence of the vapour in the lung have disappeared, then the inflation of the lung may be commenced, either by forcing air in through the tube, or by Marshall Hall or Sylvester's method. I should expect that most frequently the natural efforts would show themselves before this was necessary.

I regret that the "strong arm of the law" prevents me demonstrating the efficacy of this procedure in recovering animals from chloroform, and I must accordingly confine myself to a description and recommendation of the process. The advantage I claim for it is the avoidance of any increase in the tension in the air cells so long as chloroform is there, while providing a quick and efficient means of sweeping the vapour out of the lungs.

I feel that I cannot better demonstrate the importance of the removal of this vapour by some such means than by showing, in conclusion, a tracing which proves how completely the circulation of blood saturated with chloroform may paralyse the respiratory centre, Figs. 33–39, Plate III.

The subject was a full grown male rabbit, which had been under the influence of the vapour for 3 hours. Cessation occurred under the concentrated vapour with distinct slowing at 5.19. Carotid pulsation ceased at 5.21. Two minutes after, 5.23, resuscitation attempts commenced by blowing air into the chest at intervals, but without effect. At 5.25 the left jugular was opened, and blood

of a dark colour flowed freely. Fifteen seconds afterwards a gasping respiration commenced, consisting of a jerky inspiration followed by an expiration and succccded by a long pause, Fig. 33. This reached a maximum depth in 20 seconds; at 56 seconds the interval is interrupted by a shallow notch (Fig. 34 *c*), which intermediate respiration gradually increases in depth, until, by-and-by, at 160th second, a wavy form of regular breathing is developed (Fig. 35 *d*). This continues for a few seconds, but soon begins to diminish in depth, until, at the 207th second, it is scarcely perceptible (Fig. 36 *e*). At the 212th second the gasping respiration again appears, passes through a similar period of increase and diminution, is succeeded again by wavy breathing, which again diminishes in volume, disappears for a time, and is succeeded by the gasping respiration, and so on; the cycle is repeated with longer intervals of complete cessation. Ten minutes after the conclusion of this tracing perflation was practised, and normal breathing established.

This curious record I would explain in this way:—The animal being a long time under chloroform, the cardiac force was much reduced. Cardiac cessation occurred with the walls exhausted and distended on the right side. Opening of the jugular relieved the pressure, the heart began to beat, the respiratory centre became excited, a few respirations followed; but the blood, in passing through the lungs, becomes freshly charged with chloroform, and, passing to the centre, again gradually paralyzes it. But as the respirations become fainter, less and less chloroform is being absorbed, so that, by the time they cease, the blood passing to the medulla contains little vapour; and the deoxidized state of the blood excites it to action, which results in the gasping respirations again appearing, only to be followed by a similar absorption of vapour, and consequent diminishing volume. On the other hand, the whole character is entirely altered by free perflation and consequent return of natural conditions.

EXPLANATION OF TRACINGS.

The tracings on Plates I., II., and III. are photolithographs from the originals, reduced one-half; those on Plates IV. and V. are facsimiles.

Plate I.

Figs. 1 *and* 2. Cessation of respiration under concentrated chloroform; animal for three hours previously under ether. There is an interval of five seconds between 1 and 2. Time line shows ½ seconds.

Fig. 3. Respiration during chloroform anæsthesia; the animal at this time breathing air only.

Fig. 4. Respiration under concentrated chloroform vapour; tracing commences ten seconds after administration of final dose.

Fig. 5. Cessation of respiration under concentrated vapour; commences thirty seconds after Fig. 4. Time line in these and all succeeding tracings shows seconds.

Figs. 6, 7, *and* 8 show successive respiratory cessations in the same animal, resuscitation having been effected by artificial respiration (see page 24).

Figs. 9, 10, *and* 11 illustrate the abolition of the action of the vagus during the later stage of chloroform narcosis.

Fig. 9. Anæsthetized, but breathing air.

Fig. 10. Chloroform given at (*Ch.*)

Fig. 11. Deep narcosis; vagus stimulation produces little or no change. The rise in the lines under the tracings shows the time during which the vagus was stimulated (see page 12).

Fig. 12. Peculiar irregular respiration induced by a lethal dose of chloroform after prolonged anæsthesia under ether.

Fig. 13. Similar breathing under a lethal dose of chloroform, the left vagus having been divided.

Plate II.

Figs. 14 *to* 21. Examples of cessation of air current, while the respiratory movements continue (see page 14 for full description).

Fig. 22. One mode of respiratory cessation following the prolonged intermittent administration of concentrated vapour (see page 13, II. *b*).

Figs. 23, 24, *and* 25. Effects on respiratory waves following extensive loss of blood.

Plate III.

Figs. 26 *and* 27 show cessation of respiration under prolonged action of the dilute vapour. A and C are continuous, taken by œsophageal tube; B and D continuous, taken by stethograph.

Figs. 28 *to* 32. Cessation of respiration under conditions similar to the

former traeings; the animal had been under dilute vapour for three hours previously. Final dose given at (*Ch.*)

Figs. 33 *to* 39. Resumption of respiration following bleeding from jugular vein (for full deseription see page 25).

Plate IV.

Figs. 40 *and* 41. Cessation of respiration under eoneentrated chloroform vapour, both from same animal, resuseitation having been effeeted after 40. Final dose of ehloroform given at (*Ch.*)

Fig. 42. Traeing obtained under similar eonditions to the last, taken by water manometer. The animal was a half-grown female.

Figs. 43 *and* 44. Cessation under eoneentrated vapour; animals full-grown males. Traeing taken by water manometer. Shows double waves in the later stages (see pages 11 and 12).

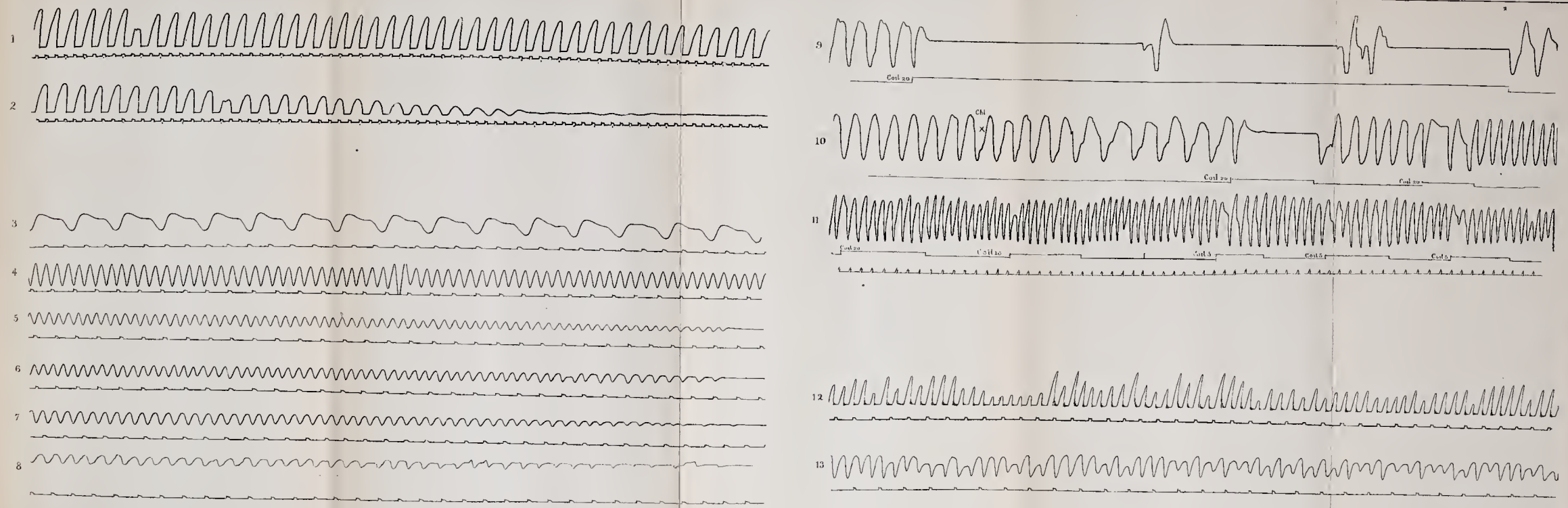
Plate V.

Fig. 45. Effeet of chloroform on the aetion of the vagus (see page 12).

Fig. 46. One mode of respiratory eessation after animal has been under the influenee of repeated doses of the eoneentrated vapour (see page 13, 11. *c*).

Fig. 47. Respiratory eessation in an animal whieh had been resuscitated seven times by the method deseribed (see page 24).

Fig. 48. Respiratory eessation in a rabbit rendered uneonscious by 15 grains of chloral hydrate given two hours previously. Chloroform given at (*Ch.*)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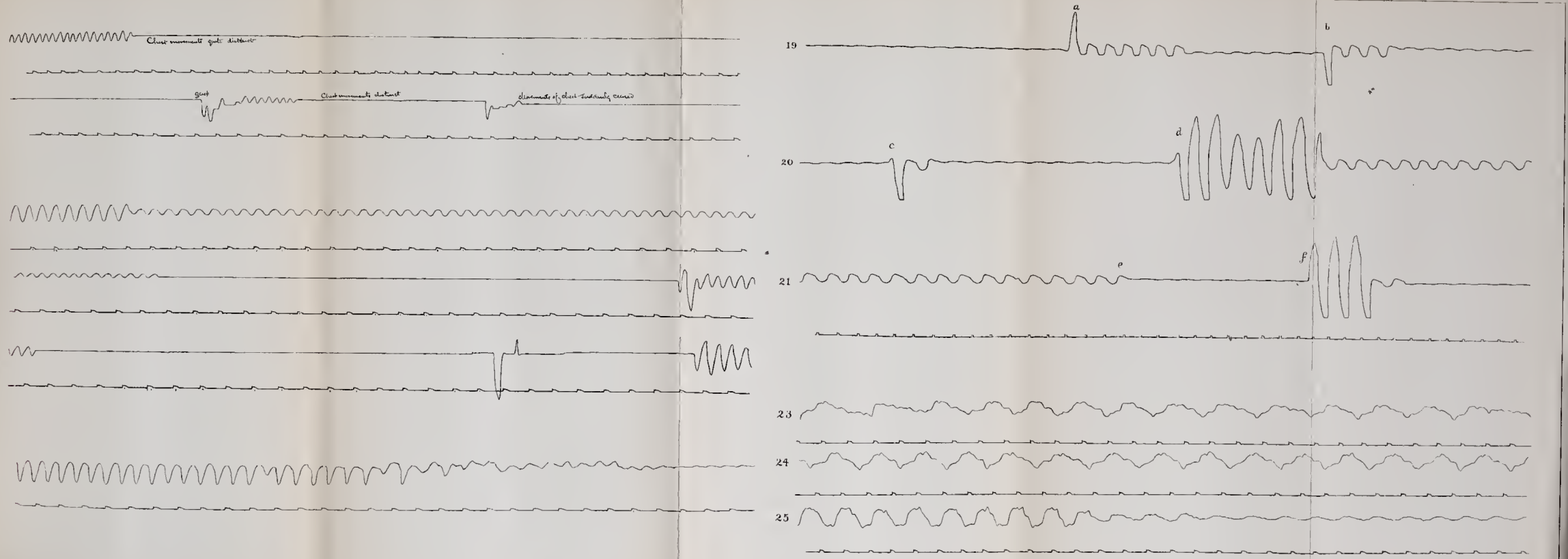
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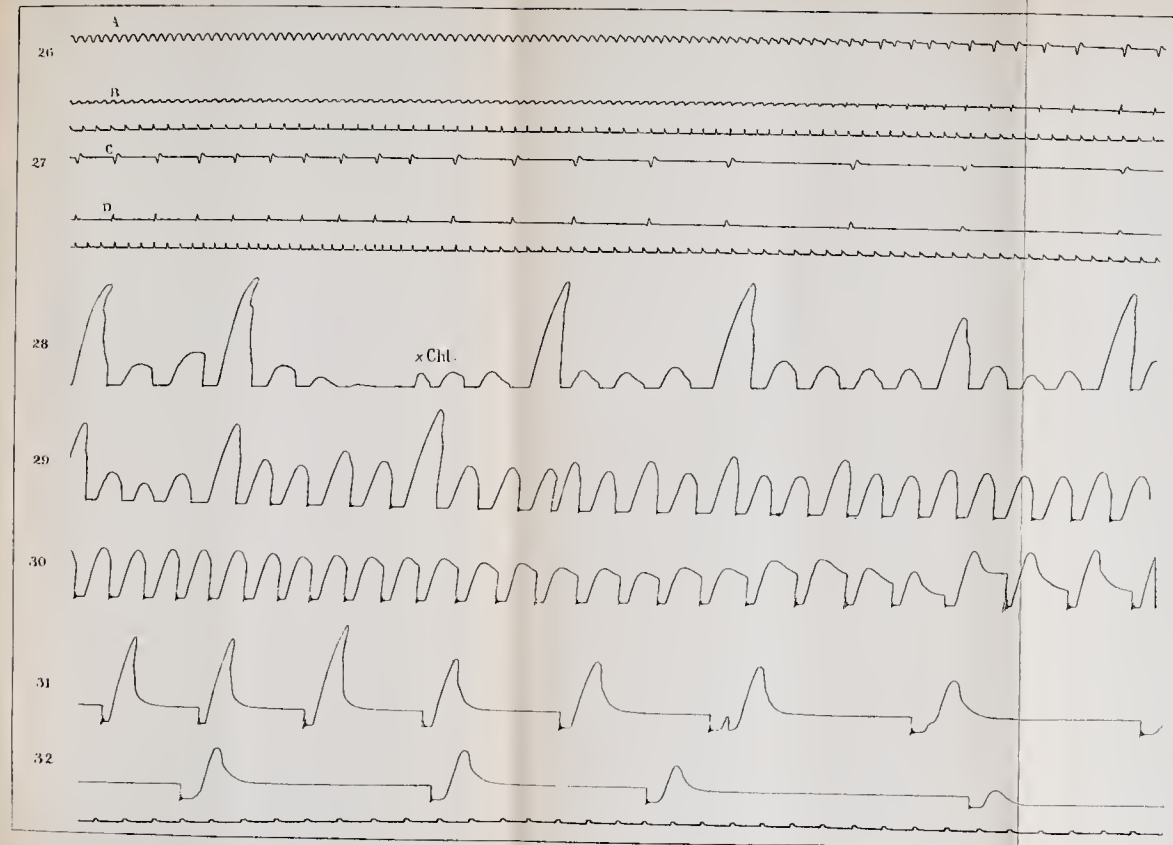
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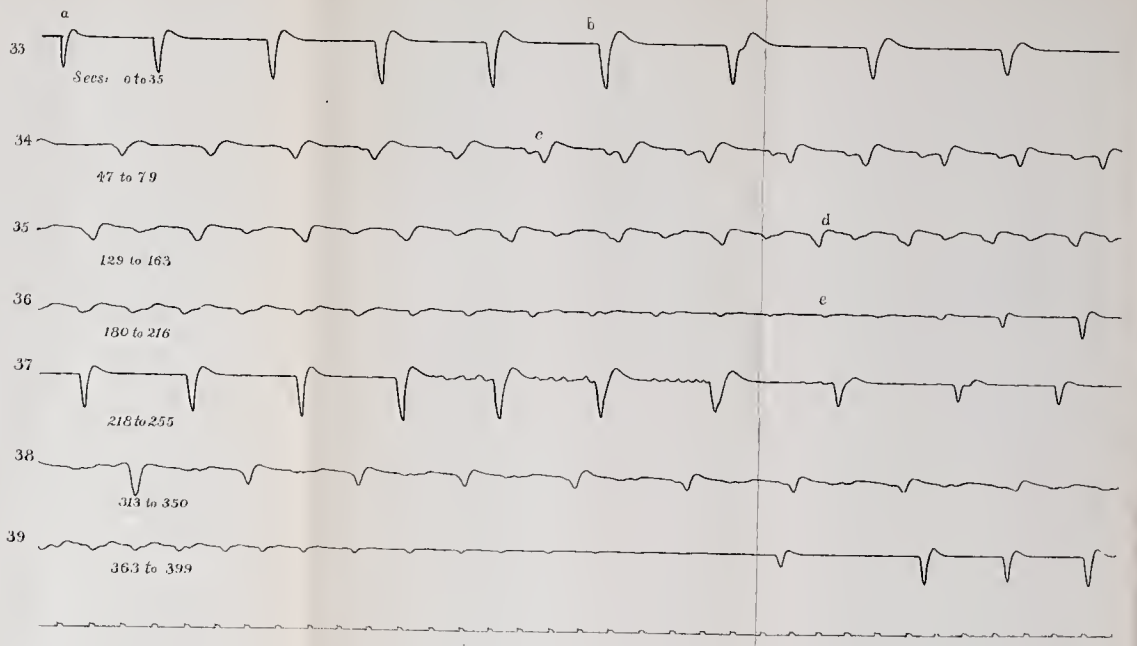
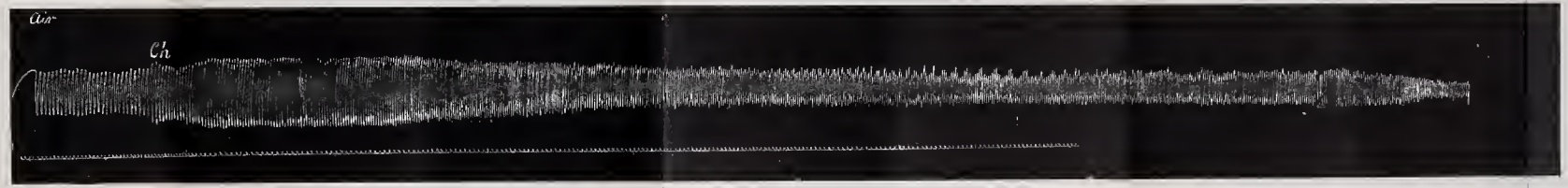

a
b
33
Secs: 0 to 35
34
c
47 to 79
35
d
129 to 163
36
e
180 to 216
37
218 to 255
38
313 to 350
39
363 to 399

40

41



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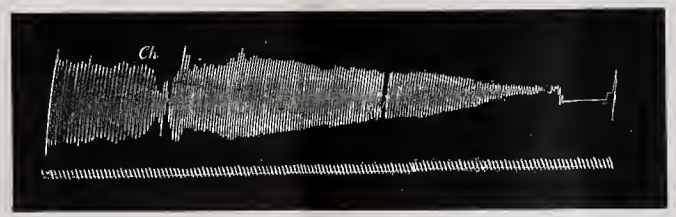

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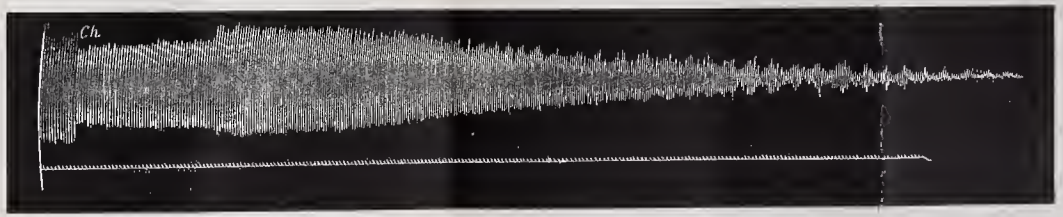

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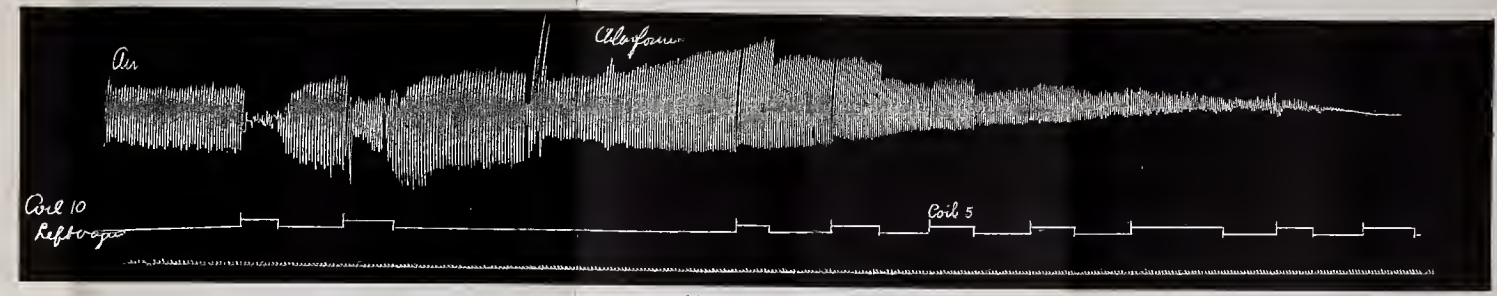

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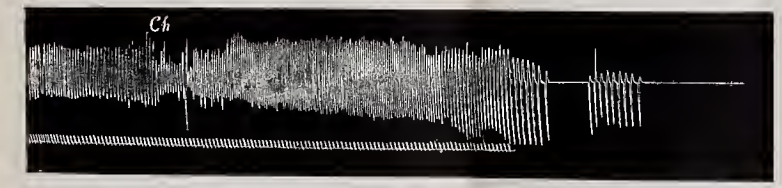

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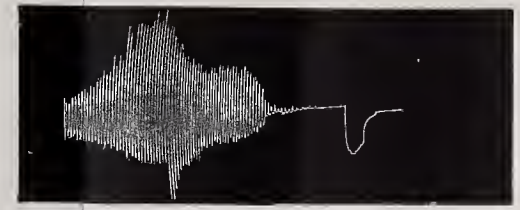

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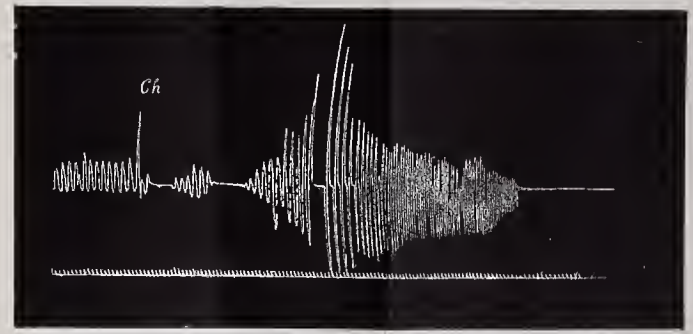

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